is relatively infrequent, and the numbers available for analysis so small as not to repay the labour of extraction.

The duration of mammary cancer is markedly longer than that of any other cancer, while that of rectal cancer comes easily second. It is interesting to note that in carcinoma of the cervix radium treatment seems to prolong life by an average of six months, thus confirming the general opinion amongst surgeons that it is of service in these cases. Only five cases of cancer of the breast were treated in this way, but of twenty-eight cases treated by x rays the average duration was only twenty-four months (against thirty-nine months for untreated cases), and each of the age periods showed a corresponding deficit.

The section of the table which deals with the thyroid gland contains, it will be noticed, bracketed figures; these include one case the duration of which was stated to be thirty years. The patient eventually died in the Cancer Hospital and the malignant nature of the growth was proved, but I find it difficult to believe that it was originally malignant, and consider it probable that malignant change ensued in a previously innocent tumour.

The excess of twenty in the total cases of cancer of the cervix over the combined totals treated by palliative measures and by radium is due to the fact that the former number contains twenty cases which received x -ray or combined x -ray and radium treatment.

Although the figures given by Lazarus-Barlow and Leeming lend support to the idea that cancer is more malignant in younger than in older persons, my figures afford no confirmation for such an opinion, and the duration of life appears fairly uniform throughout all the age periods.

For those sites which may be affected in either sex my figures give little, if any, indication that the duration of life is greater in females than in males.

TABLE III.—*Oesophagus, Larynx, Thyroid, Rectum.*

	Males.		Females.	
	No. of Cases.	Duration in Months.	No. of Cases.	Duration in Months.
Oesophagus ...	171	9	36	12
Larynx ...	44	14	8	14
Thyroid ...	4	17	9	8*
Rectum ...	256	28	194	29

* If the case mentioned in the text, and said to have had a duration of thirty years, be included, this figure becomes 43. There was no case in the series of malignant cervical glands in a female.

In conclusion, my thanks are due to Miss Hewer of the Cancer Hospital, by whom was performed the labour of searching the hospital records and abstracting the notes of over 2,000 cases, which form the basis of these statistics.

THE USE OF INTARVIN IN DIABETES MELLITUS.

BY

D. MURRAY LYON, M.D., W. ROBSON, PH.D.,
AND A. C. WHITE, M.B.

(From the Department of Therapeutics, University of Edinburgh.)

THAT the fatty acids are broken down during oxidation by the removal of two terminal carbon atoms at a time (β -oxidation) seems to be fairly well established. Knoop¹ in 1904, in his classical investigations on the fate of the phenyl fatty acids in the animal organism, was the first to propound this theory, which was ridiculed by the pure chemists until Dakin² was able to show that an analogous breakdown of these and similar substances could take place *in vitro*. The investigations here referred to were all concerned with the oxidation of fats with an *even* number of carbon atoms in their molecule. Höckendorf³ had earlier shown that the introduction into the diabetic

organism of alcohols containing an *odd* number of carbon atoms, and which therefore on oxidation probably give rise to fatty acids with an uneven number of carbon atoms, brought about an increased excretion of glucose. Embden and his co-workers⁴ found that on perfusing freshly excised liver with the lower members of the fatty acid series only those containing an *even* number of carbon atoms in their molecule yielded aceto-acetic acid as a result of their katabolism. Then Ringer and Jonas,⁵ working with homologues of propionic acid, showed that valeric and hepatic acids yielded glucose in the diabetic organism roughly in proportion to the amount of propionic acid that would be found as the result of β -oxidation, while butyric and caproic acids led to the increased excretion of aceto-acetic acid without increased glucose elimination. It was then that Ringer conceived the idea of looking for a suitable odd-carbon atom fat to be used in the treatment of acidosis, and which could be given in considerable amounts to diabetics without the possibility or danger of acidosis arising. His attempts, however, to synthesize such a suitable fat were unsuccessful. Other writers—namely, Janeway and Mosenthal—also saw the possibilities. Max Kahn succeeded in synthesizing a suitable fat— $C_{16}H_{33}COOH$, margaric acid—which is easily purified and which, when united with glycerol, forms a neutral fat. This compound, commercially known as intarvin, apparently can be katabolized in the organism without yielding ketogenic derivatives. Kahn states⁶ that on administration to phlorizinized dogs this fat causes a marked increase in glucose elimination in the urine, showing that in the dog it is broken down to propionic acid and then converted into glucose in accordance with the theory brought forward by Ringer and his collaborators. In this communication he brings forward a certain amount of data to confirm his claims of the previous year.⁷ He recommends the use of this fat in those cases where insulin has no apparent or only a moderate effect up to the extent of 1,000 calories daily from such a source. He also gives data from a patient with diabetes complicated by tuberculosis, who was fed with intarvin. In this subject it was found that 100 grams of intarvin given in addition to the ordinary diabetic diet caused no ketonuria, and the carbon dioxide combining power of the blood increased, while the blood sugar during this time remained below the threshold value. In December, 1923, he published⁸ a more extensive series of observations to substantiate his claim.

In his first case, one of starvation acidosis, he found that the addition of 100 grams of intarvin caused the acetone to disappear, while on replacing it the next day with an equal weight of butter the acetone returned once more, to disappear next day on administration of the same quantity of intarvin.

In his second case, showing acetoneuria and acetonæmia even on a low fat diet, the addition of 90 grams of intarvin cleared the acetoneuria, and when an aliquot portion of intarvin was given the next day the acetonæmia also went. The patient's blood sugar also came nearer to a normal level.

In the third case it was found that when the patient had been rendered ketone-free without intarvin, 150 grams of this substance could be added to his diet without ketonuria reappearing, and the bicarbonate reserve of the blood increased while the blood sugar remained normal.

In the fourth case, one of pituitary glycosuria where insulin was ineffectual, feeding with the odd-carbon fat cleared up the acetone and brought about a gain in weight and the glycosuria was controlled. When the intarvin was omitted, ketonuria and glycosuria immediately reappeared.

Joslin⁹ also reports favourably upon this fat. He states:

"It would appear to be ideal in the instance of acidosis in the presence of infection"; and "It can logically be recommended to those cases with whom the fat-carbohydrate ratio is above 3:1."

French workers¹⁰ have also brought forward evidence in favour of the antiketogenic function of intarvin. There are, however, certain dissentients. Sevringhaus¹¹ reports that after comparing the effect of ingestion of beef suet and an equal quantity of glyceryl margarate in a ketonuria following carbohydrate starvation—

"Glyceryl margarate seems to have little or no ketolytic value aside from its glycerol content. It does not exert a protein sparing action in these diets."

Allen¹² condemns the use of artificial foodstuffs, including intarvin, in endeavouring to provide weight and strength for the diabetic, as for ever fruitless, being based on a superficial and false conception of the disease.

In view of this it seemed of interest to see what could be done with intarvin in cases of diabetes mellitus under treatment. Some of the relationships of intarvin to metabolism were consequently determined in four diabetic subjects, and, in addition, the effect of the absorption of intarvin on the blood fats was determined in two normal individuals.

Regarding the palatability of the fat, individual taste varies, but in our experience it was always found to be disagreeable, more especially if taken along with other food. Probably the mineral oil with which it is mixed when prepared for commerce had something to do with this. There is, however, no doubt that the intarvin is absorbed; both Kahn and Sevringhaus (loc. cit.) showed it to be 97 per cent. absorbed—that is, practically as well as beef suet would be under similar conditions. Considering that it was supposed to be antiketogenic in function it was thought worth while to determine whether the absorption of intarvin had any effect on the blood sugar.

Intarvin in Normal Subjects.

The following table shows the response of one of the normals (W. A.) to 250 c.cm. of 40 per cent. cream on a fasting stomach.

TABLE I.—Response to Ingestion of 250 c.cm. of 40 per cent. Cream. (Normal Subject.)

	Hours after Ingestion.					
	0	1	2	3½	5	6½
Total blood fat in mg. per cent. (Blood)	455	435	411	504	460	414
Blood sugar in mg. per cent. ...	97	106	97	101	111	—
Respiratory quotient ...	0.90	0.80	0.78	0.79	0.81	0.80

Figures for cholesterol (whole blood) and for the metabolic rate showed no significant change and are not recorded. The maximum rise in the blood fat occurs earlier than in the cases reported by Cowie and Hoag,¹³ otherwise there is little to note. The intarvin absorption curve of this subject shows similar features. On one occasion the intarvin, to render it more palatable, was made up with washed bran. The blood cholesterol showed a considerable rise at the end of the first hour (accountable for by the sterol content of the bran itself). Details being more complete as regards the intarvin absorption curve of the other normal (W. R.), the results are given in Table II.

TABLE II.—Response to 100 grams of Intarvin. (Normal Subject.)

	Hours after Ingestion.					
	0	1	2	3½	5	6½
Total bloodfat in mg. per cent. (Blood)	770	806	881	916	823	818
Blood sugar in mg. per cent. ...	103	125	118	114	108	125
Respiratory quotient ...	0.76	0.74	0.79	0.79	0.81	0.82

Blood cholesterol and metabolism showed no significant changes. In both cases it was found that the ingestion of the intarvin resulted at the end of the experiment in a certain looseness of the bowels, due probably to the mineral oil it contains. At no time did the respiratory quotient give any indication of the burning of carbohydrate in excess of normal. The urine in both cases did not show any abnormal constituents.

Intarvin in Diabetic Subjects.

In order to determine accurately the value of intarvin in diabetes it was considered desirable to have the diets of the patients stabilized at the highest possible level within the limit of tolerance, and then to add a quantity of intarvin. Patients were treated on the lines described in the *BRITISH MEDICAL JOURNAL*, August 23rd, 1924.¹⁴ The diabetic cases were as follows:

CASE I.

A. R., aged 31. History of diabetes for four months. Admitted February 20th, 1924. On a diet of 1,240 calories ("B 12": carbohydrate 64.5 grams, protein 47.5 grams, fat 89 grams. G:FA::1:1) the patient excreted 41 grams of glucose in twenty-four hours. Acetone positive in the urine. The acetone and sugar were both got rid of from the urine by dieting alone, and the diet was built up to 2,400 calories. On March 12th on this diet it was found that the blood acetone (Van Slyke and Fitz) rose to 100 mg. per cent. two hours after breakfast, falling to 9 mg. per cent. two hours later, and remaining at this level for another two hours. Next day, similar conditions showed a fasting blood acetone of 5.8 mg. per cent., rising to 640 mg. two hours after a breakfast to which 100 grams of intarvin had been added, and falling to 36 mg. per cent. two hours later, to rise at the fourth hour to 42 mg. The blood fats showed a certain parallelism, but rose again at the end of the period. There was definite acetonuria, 364 mg. per cent. of acetone being present in the urine passed four hours after the intarvin.

CASE II.

W. D., a gardener, aged 41. Duration of diabetes uncertain. An ophthalmologist diagnosed diabetes three months before admission, while the sight of the right eye had begun to fail twelve months before that. Admitted March 18th, 1924, on a diet of 1,524 calories ("B 15": carbohydrate 78 grams, protein 60 grams, fat 108 grams. G:FA::1:1). On March 19th the urine was acetone-free and the total glucose lost in twenty-four hours was 23.4 grams. The diet was gradually and with difficulty built up to 1,505 calories ("C 15": carbohydrate 50 grams, protein 49.5 grams, fat 123 grams. G:FA::1:1.6). Blood examinations were made during April 10th; these were repeated on April 11th, 100 grams of intarvin having been given in addition to the breakfast. The intarvin was continued at the rate of 100 grams per diem on the same diet until April 16th. The figures for April 17th represent the termination of the intarvin period. The specimens recorded as before 10 a.m. are fasting samples. Details of examinations in this case are given in Table III.

TABLE III.—CASE II.

(All chemical data are given in milligrams per cent.)

Date and Time.	Blood.			R.Q.	Urine.	
	Sugar.	Total Fat.	Acetone.		Sugar.	Acetone.
April 10—						
9.30 a.m. ...	222	809	12.8	0.79	—	8.9
10 a.m., breakfast.						
12 noon ...	222	835	10.6	0.80	—	—
4.30 p.m. ...	200	894	14.8	9.92	0.5%	26.7
*April 11—						
9.5 a.m. ...	200	813	10.6	0.91	—	29.4
12 noon ...	232	913	10.6	0.73	—	—
4.30 p.m. ...	221	769	10.6	0.75	—	46.0
*April 12—9.30 a.m. ...	265	1114	17.0	—	—	46.7
*April 13—9.30 a.m. ...	265	1110	12.8	—	—	28.2
*April 14—9.30 a.m. ...	210	1072	27.7	0.91	—	20.2
*April 17—9 a.m. ...	205	783	47.4	0.81	—	30.3

* Indicates days during which intarvin was given.

The twenty-four hour urine during the intarvin period showed a slight trace of sugar, not estimable. Cholesterol and general metabolism figures revealed nothing. During the period of intarvin administration there was a temporary rise in the fasting blood fat and a gradual rise in the blood acetone percentage, fairly well reflected in the findings for urinary acetone when compared against the fasting blood samples. The respiratory quotient showed no typical change. The faeces during the intarvin period contained 47 per cent. fat, of which 84.5 per cent. was unsplit. The large percentage of unsplit fat was probably due to the unabsorbed mineral oil portion of the intarvin.

CASE III.

D. B., aged 30. Duration of diabetes, fifteen months. On April 19th, 1924, the patient was put on a diet of 1,524 calories ("B 15": carbohydrate 78 grams, protein 60 grams, fat 108 grams. G:FA::1:1). The urine showed acetone, and 41.1 grams of glucose were lost in the twenty-four hours on this diet. The patient was made sugar- and acetone-free with difficulty. The diet was built up to 1,896 calories ("C 19": carbohydrate 61 grams, protein 57.5 grams, fat 158 grams. G:FA::1:1.5). With the diet at this level on May 20th, from May 21st to May 29th the same diet was given, with, in addition, 100 grams of intarvin per diem. During this period the blood acetone was only slightly increased, and the total fats and cholesterol remained fairly steady except towards the end, when the blood fat started to rise. The cause of this cannot be explained. Later, when the patient

TABLE IV.—CASE IV.

Date and Time.	Diet.				Blood.			R.Q.	Morning Urine.		24 Hours' Urine.	
	C.	P.	F.	Intarvin.	Sugar.	Total Fat.	Acetone.		Sugar.	Acetone.	Sugar.	Acetone.
	gm.	gm.	gm.	gm.	mg. %	mg. %	mg. %		mg. %	mg. %	gm.	gm.
A. Intarvin and Pre-stabilization Period.												
July 16, 1924—												
10.5 a.m.	25.0	37.75	50.5	0	125	—	16	—				
12.12 p.m.					118	861	87	—				
4.15 p.m.					114	1065	—	0.88				0.15
July 17—												
9.30 a.m.	31.5	39.75	72.0	100	114	926	52	0.77				
12.15 p.m.					111	935	215	0.77				
1.35 p.m.					—	932	169	—				
4.0 p.m.					138	957	61	0.77				0.69
B. Intarvin added to Stable Diet.												
August 6.—9 a.m.	81.0	69.5	200.0	100	—	1054	11	0.73	—	9	—	—
„ 7.—9 a.m.	81.0	69.5	200.0	100	125	1047	6	0.70	—	15	Sl. trace	—
„ 8.—9 a.m.	81.0	69.5	200.0	100	108	1127	8	0.73	—	—	Trace	—
„ 9.—9 a.m.	81.0	69.5	200.0	100	104	1235	64	0.74	—	11	Trace	0.15
„ 10.—9 a.m.	36.0	29.0	77.0	100	—	—	—	—	—	—	Trace	—
„ 11.—9 a.m.	77.6	69.0	190.5	100	105	1264	75	—	—	27	Trace	0.87
„ 12.—9 a.m.	77.6	69.0	190.5	100	110	871	45	0.78	—	15	Trace	0.71
C. Fat added to Stable Diet.												
August 14.—9 a.m.	77.6	69.0	240.5	*	100	1034	83	0.75	—	—	—	—
„ 15.—9 a.m.	77.6	69.0	240.5	*	111	1052	149	0.76	—	107	—	—
„ 16.—9 a.m.	81.0	69.5	200.0	*	91	956	57	0.70	—	169	Sl. trace	—
„ 21.—9 a.m.	77.6	69.0	273.5	†	—	—	—	—	—	—	—	—
„ 22.—9 a.m.	77.6	69.0	190.5		97	893	86	0.66	—	151	—	1.5

Note.—In sections B and C above the figures are obtained from samples of blood taken, and of urine passed about 9 o'clock each morning while the patient was still fasting.

* In section C 50 grams of butter fat replaced the intarvin previously given. † 100 grams olive oil added to diet of August 21st.

was put on insulin 20 units per diem, the fats rose still higher. The total urinary acetone for twenty-four hours was on May 21st 0.35 gram, and on May 28th 1.35 grams. There was a faint indeterminate trace of lower sugar per twenty-four hours during most of the intarvin period. During the whole of the period the fasting blood sugar was below the normal threshold value. Thus, while the evidence obtainable for blood acetone was doubtful, we found that the total acetone excretion had risen. As in Case II, the faeces showed a high percentage of unsplit fat during intarvin administration, probably for the same reason.

CASE IV.

D. R., aged 55. Diabetes discovered four years previously during examination for life insurance. Apparently the disease had always been of the mild type. The patient was admitted on July 10th, 1924. After a test meal of 50 grams of glucose the blood sugar rose to 200 mg. per cent. at the end of the first hour, but had returned to normal at the end of three hours. In this subject the intarvin was administered in two different periods: (a) in the pre-stabilization period as the diet was being built up (July 16th and 17th); (b) from August 6th, when the diet was in a more stable condition. During the first period it was found that the addition of intarvin to the diet was associated with increase of acetone.

The diet was then built up without intarvin (to "C 25"—that is, 2,520 calories per diem), but to make sure that even with intarvin the diet would still be within the patient's tolerance it was kept 100 calories lower during the experiment—that is, 100 grams of intarvin were added to a diet containing 2,400 calories from August 6th to 12th. August 10th, however, was made a low day, with only 952 calories besides the 100 grams of intarvin, and the diet on August 11th and 12th was kept at 2,300 calories along with the intarvin. With the exception of the first day, the twenty-four hours sample of urine showed an indeterminate trace of sugar throughout the whole period. This disappeared on stopping the intarvin. The diet was kept at 2,300 calories per diem during August 14th and 15th, and 50 grams of butter fat were added each day. On August 21st 100 grams of olive oil were given along with ordinary diet, and acetoneuria was definitely produced.

It will be noticed that, even during the stabilized period, intarvin when added to the diet tended to increase the percentage of acetone in the blood and also the total excretion of acetone, especially towards the end of the period.

This, in view of its supposed antiketogenic function, is rather disappointing. The increase, however, is not so marked as when ordinary fat is given—for example, the figures of August 14th and 15th, when 50 grams of fat were given to replace the intarvin. When 100 grams of olive oil were taken on August 21st there is again a much greater increase in the total acetone lost.

Discussion.

From the above observations on the use of intarvin in diabetes it would seem that further investigations are needed into the whole question before the claims on behalf of this fat can be fully substantiated. In the cases recorded no reduction, but rather an increase, in the amount of acetone in the blood followed the administration of intarvin. The rise, however, is less than when an equivalent quantity of other fat is added to the diet. The average G: FA ratio in the diets employed was 1 to 1.5, so that there was ample carbohydrate to burn up the ordinary fat of the diet, apart from the intarvin. The respiratory quotient did not at any time give indication of more carbohydrate than usual being burned under the influence of intarvin, as might be expected if the acid ultimately broke up to form sugar. On the other hand, the addition of intarvin to a stabilized diet always led to the excretion of small traces of sugar in the urine. The changes in the blood fat under intarvin are simply recorded. These cannot be explained as yet. Further investigation of this question is in progress. When a patient's diet has been stabilized at his maximum tolerance, 100 grams of intarvin can be added (allowing about 700 extra calories) without the appearance of clinical signs of acidosis. But, in view of the conflicting figures obtained regarding the control of ketosis consequent on its use, intarvin cannot be used indiscriminately to build up diets to a maintenance level without fear of ketosis. The expense

and the unpalatability of the substance will also militate against its general acceptance in practical dietetics.

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THE PERMEABILITY OF MENINGES TO URANIN AS A METHOD IN THE DIAGNOSIS OF MENINGITIS.

(Preliminary Communication.)

BY

OTTO JERVELL, M.D.

(From the Christiania (Oslo) Municipal Hospital,
Medical Section VIII.)

A METHOD whereby it is possible to decide simply by lumbar puncture whether meningitis is present or not may possibly be of interest. The normal meninges and plexus choroides have the power of preventing a number of substances that are foreign to the organism from passing into the spinal fluid; diseased meninges have lost this power. Especially in meningitis this permeability of the meninges to chemical substances is greatly increased, and it is thus natural to investigate whether the determination of the latter can be of assistance in diagnostic difficulties. The substance must be easily found in the spinal fluid and be non-poisonous. Such a substance is uranin (sodium fluorescein), a red colouring matter very readily soluble in water; a weak solution gives an intensely light-greenish fluorescence which strongly recalls the fluorescence seen in a positive Schlesinger reaction. The fluorescence does not disappear until the solution is weakened to 1 in 10,000,000. Weaker solutions have a dirty greenish colour, without fluorescence.

A series of investigations carried out by Kafka,¹ Schönfeld,² Jörgensen,³ and others have established that uranin, in doses of 6 to 8 grams by the mouth, passes into the spinal fluid in about 30 per cent. of healthy individuals, but in small quantities only. In dementia paralytica, and also in syphilitic persons, uranin was found more frequently in the spinal fluid, especially if it was altered. These authors do not appear to have made any determination of the permeability of the meninges in meningitis.

Uranin given in larger doses enters the spinal fluid where the meninges are not infected, and the patient's skin becomes intensely yellow. After several experiments we decided upon 2 grams of uranin for an adult, or about 0.03 per kilogram of body weight. One or two hours after such a dose the patient turns yellow, but the coloration is not stronger than in icterus, and the yellow colour of the skin and mucous membrane disappears comparatively quickly, as a rule after five to six hours. Uranin has no poisonous effects, and according to C. Jörgensen's investigations does not irritate the meninges. It can be administered by the mouth or by intramuscular injection in a 20 per cent. solution, but not subcutaneously, as a strong solution produces a sensitive infiltrate. After half an hour to an hour the blood serum gives a greenish fluorescence, which in the great majority of cases does not disappear until the serum is weakened to 1 in 64. The concentration of uranin in the blood is strongest two to three hours after it has been given, and it is at that time, therefore, that the lumbar puncture should be performed. We employ the following technique:

The patient is given 2 grams of uranin by the mouth (preferably in wafers) or by intramuscular injection (20 per cent. solution) if he is not quite conscious. Three hours later lumbar puncture is undertaken in the usual manner. In order to ascertain whether

the spinal fluid is fluorescent or not, the test tube must be held against a dark background. In meningitis the fluid gives off a distinct greenish fluorescence. No importance should be attached to a doubtful fluorescence. The presence of blood may confuse the reaction, since the blood contains great quantities of uranin. Investigations made at this section prove, however, that at least 1/3 c.cm. of blood must be mixed with 10 c.cm. of spinal fluid in order that the latter shall become fluorescent.

We have employed this simple method with 74 patients, 18 of whom were suffering from various forms of meningitis. The spinal fluid (from, in all, 22 lumbar punctures) taken from patients suffering from meningitis was in all cases fluorescent, in some quite strongly, like a markedly positive Schlesinger reaction. It is true that the fluorescence was weak in three instances, but nevertheless distinct. The method used in these cases, however, differed somewhat from the technique usually employed, the lumbar puncture being performed within one hour after the patient had received the uranin. In addition, the dose of uranin given to two of the patients was too small (1 gram). The spinal fluid in 38 of 56 non-meningitic cases was as clear as water. In 8 cases the fluid was of a dirty green colour without fluorescence; this shows that minimum quantities of uranin had entered the spinal fluid. In 10 cases the spinal fluid was faintly fluorescent, but in 7 of these there was an admixture of blood, which, as mentioned above, makes the results uncertain. Three of the non-meningitic cases, on the other hand, exhibited a faintly fluorescent spinal fluid three hours after 2 grams of uranin. The fluorescence disappeared, however, in a dilution of 1 in 2, whilst the spinal fluid from patients with meningitis showed fluorescence in a dilution of 1 in 8 and up to 1 in 30. (For this determination we employed small test tubes 11 mm. in width, and observed the fluorescence in a dark box, where the light at a distance of 15 cm. enters through a 20 dioptric biconvex lens.)

Conclusions.

Uranin in doses of 0.03 gram per kilo of body weight does not enter the cerebro-spinal fluid, or does so only in small quantities if the meninges are not infected. In meningitis the colouring matter enters the fluid in easily demonstrable quantities. If, therefore, by lumbar puncture three hours after uranin has been given in the doses stated, the spinal fluid shows a distinct greenish fluorescence, we can with great probability diagnose meningitis without making other investigations of the spinal fluid. If the spinal fluid shows no fluorescence, meningitis can with certainty be excluded.

It is not possible to decide what form of meningitis, if any, is present. The passing of the colouring matter into the spinal fluid merely indicates the greater or lesser permeability of the meninges. In meningitis comitans, circumscripta, etc., the uranin does not enter the fluid, or does so in relatively small quantities, even when pleocytosis is present. Opinions differ as to whether it is correct to designate these conditions meningitis, but this is doubtless chiefly a dispute about words.

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A WEAK POINT IN STERILIZING METHODS.*

BY

KENNETH BLACK, F.R.C.S.ENG.,

PROFESSOR OF SURGERY, KING EDWARD VII COLLEGE OF MEDICINE,
SINGAPORE.

ONE of the problems of the operating theatre is to ascertain the temperature reached in the centre of a packet, tin, or drum of dressings in a sterilizer. It is very different from that of the steam around the outer side of the packet or tin, but the latter alone is registered by a thermometer projecting into the sterilizer and capable of being read from the outside, or, more commonly, by means of a pressure gauge. I made tests with a specially prepared thermometer, similar to a clinical thermometer

* Read at a meeting of the Malaya Branch of the British Medical Association.